Review

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Clinical significance of antibodies to antigens in the International Society of Blood Transfusion collections, 700 series of low-incidence antigens, and 901 series of high-incidence antigens

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This article reviews information regarding the clinical significance of antibodies to antigens in the blood group collections, the 700 series of low-incidence antigens, and the 901 series of high-incidence antigens. Antibodies to many of the antigens in these groups are rarely encountered, meaning that available information is limited. For a few, the clinical significance—the potential to cause reduced survival of transfused antigen-positive red blood cells, a hemolytic transfusion reaction (e.g., anti-AnWj, anti-Emm), or hemolytic disease of the fetus and newborn (e.g., anti-Kg, anti-HJK)—has been documented. Many other specificities have so far been benign (e.g., anti-Cs^a, anti-M_1).
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When an antibody, directed at an antigen expressed on red blood cells (RBCs), is present in the plasma of a patient who requires transfusion, the clinical significance of the antibody must be assessed to determine if blood lacking the corresponding antigen should be selected for transfusion.[1] In general, an antibody is considered to have clinical relevance if it has been associated with acute or delayed hemolytic transfusion reactions (HTRs) or with a notable shortening in the survival of transfused RBCs or if it has caused hemolytic disease (or anemia) of the fetus and newborn (HDFN). Information relating to the clinical significance is based on previous experience with a particular antibody specificity. Thus, correct identification of the antibody specificity is essential. Although the need to identify the antibody specificity is obvious, at times identifying the (correct) antibody specificity may not be a simple task.

Antigens that are authenticated by the International Society of Blood Transfusion (ISBT) Working Party for Red Cell Immunogenetics and Blood Group Terminology fall into one of four classifications: blood group systems, collections, series of low-incidence antigens, and series of high-incidence antigens.[2] This review summarizes relevant information about the clinical significance of antibodies to antigens in the ISBT collections (the 200 series), the 700 series of low-incidence antigens, and the 901 series of high-incidence antigens. In the interest of space, few original references are cited. The reader wishing for more details is referred to Daniels[3] and Reid et al.[4]

Collections (ISBT 200 Series)

A collection consists of two or more antigens that are related serologically, biochemically, or genetically but that do not fit the criteria required for blood group system status. A blood group system is defined as consisting of one or more antigens controlled at a single gene locus or by two or more very closely linked homologous genes with little or no observable recombination between them. Table 1 shows information for the six currently established collections: Cost, Ii, ER, GLOB, "unnamed," and MN CHO (MNS carbohydrate antigens). When the antigens in a collection become assigned to a system, the collection becomes obsolete; there are now seven obsolete collections: 201 (Gerbich), 202 (Cromer), 203 (Indian), 204 (Auberger), 206 (Gregory), 211 (Wright), and 212 (Vel).[2]

Cost Blood Group Collection: Anti-Cs^a and Anti-Cs^b

This collection contains two antigens: Cs^a and Cs^b. Cs^a has a high prevalence as the antigen is expressed on RBCs of more than 98 percent of people in most populations; in black

Table 1. ISBT collections (200 series) and the antigens they contain

Collection			Antigen			
Number	Name	Symbol	Number	Symbol	Prevalence (%)	
205	Cost	Cost	205001	Cs[a]	95	
			205002	Cs[b]	34	
207	Ii	I	207002	I	*	
208	Er	ER	208001	Er[a]	>99	
			208002	Er[b]	>1	
			208003	ER3	>99	
209	Globoside	GLOB	209003	LKE	98	
210	"Unnamed"		210001	Le[c†]	1	
			210002	Le[d†]	6	
					Prevalence (%)	
					Blacks	Whites
213	…	MN CHO	213001	Hu	7–22	1
			213002	M_1	16.5	0.5
			213003	Tm	31	25
			213004	Can	60	27
			213005	Sext	24 (N+)	0
			213006	Sj	4	2

*All RBCs have trace amounts of i antigen, but by standard tests i may appear to be of low incidence.

†Anti-Le[c] and anti-Le[d] react with Le(a–b–) RBCs from non-secretors and secretors, respectively.

ISBT = International Society of Blood Transfusion.

populations, the prevalence is slightly lower (95%). Cs[b] is polymorphic (prevalence of 34%). Only one example of anti-Cs[b] (an IgG antibody) has been reported; thus, there is no information regarding its clinical significance.

Anti-Cs[a] is an IgG antibody that is detected by antiglobulin methods but has not been implicated in transfusion reactions or HDFN. Although not clinically significant, anti-Cs[a] may cause a delay in transfusion because the specificity can be difficult to identify. Anti-Cs[a] reactivity has many of the features of antibodies to Knops blood group antigens; indeed, five of the original Cost antigens are now part of the Knops system. The challenges of working with anti-Cs[a] include that many are weakly reacting antibodies that are mostly found in patients with multiple antibodies. Furthermore, the Cs[a] antigen has variable expression on RBCs from different people; thus, the reactivity pattern may be confusing. There appears to be a phenotypic association between Cs[a] and Yk[a], as RBCs of approximately 12 percent of whites and 15 percent of blacks with the Yk(a–) phenotype are also Cs(a–). Because Cs[a] is one of the antigens that is suppressed on RBCs with the dominant Lu(a–b–) phenotype [encoded by *In(Lu)*], it is perhaps not too surprising that anti-Cs[a] has on occasion initially been misidentified as anti-AnWj. Anti-AnWj, in contrast to anti-Cs[a], is a specificity that has on occasion caused transfusion reactions (see later section "Anti-AnWj"). The typical recommendation for patients with anti-Cs[a] is to use the "least incompatible" (a term that has generated much discussion) RBC unit(s) for transfusion.

Ii Blood Group Collection: Anti-i

Anti-i recognizes the i antigen; i is now the only antigen in the Ii collection because the I antigen was promoted to I blood group system status.[5] The i antigen is on unbranched carbohydrate chains of repeating *N*-acetyllactosamine units on glycolipids and glycoproteins on RBCs and on proteins in plasma. All RBCs express some i antigen, and the expression is variable, with RBCs from neonates having the strongest expression. It follows, therefore, that all anti-i are autoantibodies. In healthy people, autoanti-i is rare and primarily an IgM antibody weakly reactive at lower temperatures (4–10°C). In pathological situations, autoanti-i can have a high titer and a wide thermal range, react at 37°C, bind complement, and be IgG or a mix of IgG and IgM. Autoanti-i is pathologically significant in cold agglutinin syndrome and mixed-type autoimmune hemolytic anemia. The antibody is often found in plasma from patients with infectious mononucleosis or other lymphoproliferative disorders (e.g., Hodgkin's disease) and occasionally causes hemolysis. Anti-i has been reported to occur in 64 percent of patients with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS).[6] Because all RBCs express some i antigen, i– blood is not available for transfusion. Mostly this is not an issue, although acute intravascular hemolysis occurred in a patient with anti-i after transfusion of 2 RBC units that were compatible in the immediate spin crossmatch. With regard to HDFN, maternal autoanti-i can cross the placenta and has caused mild neonatal jaundice or resulted in the RBCs of the newborn to react in the direct antiglobulin test (DAT).

ER Blood Group Collection: Anti-Er[a], Anti-Er[b], and Anti-ER3

This collection consists of two antigens of high prevalence (Er[a] and ER3) and one of low prevalence (Er[b]). The antibodies directed at these antigens are rare, so information about their clinical significance is limited. All three specificities have been found to be IgG antibodies that react by antiglobulin methods.

Anti-Er[a] has caused reduced RBC survival but no overt HTR. Clinical HDFN has not been reported, but anti-Er[a] has been associated with neonatal RBCs being positive in the DAT.

The only reported patient with anti-ER3 experienced a mild transfusion reaction. No data are available for HDFN, since the only example of anti-ER3 was made by a male patient. Because Er(a–) or ER:–3 blood is unlikely to be found, unless the patient has a compatible sibling, it is recommended by some that "serologically least incompatible" blood should be used with extra caution. Only one example of anti-Er^b is reported; therefore, its clinical significance with regard to transfusion is not known. This antibody has not caused clinical HDFN but was associated with a positive DAT.

GLOB Blood Group Collection: Anti-LKE

Anti-LKE recognizes the high-incidence antigen, LKE, now the only antigen in the GLOB collection. LKE is located on a globoside molecule that has additional galactose (Gal) and *N*-acetyl neuraminic acid residues. The expression of LKE on RBCs varies: 80–90 percent of people have RBCs with strongly expressed LKE, and 10–20 percent of people have RBCs with weakly expressed LKE. The LKE– phenotype is found in only 1–2 percent of the population. Therefore, anti-LKE is rare; only six examples have been reported, and some appear to be "non–red cell immune." They are IgM antibodies with optimal reactivity at room temperature or lower and may bind complement. One antibody was associated with posttransfusion hemolysis, but there have been no reports of HDFN. The antibody can be mistaken for anti-P as RBCs with the rare p, P_1^k, and P_2^k phenotypes do not react, and anti-LKE may be difficult to identify as RBCs typed for LKE are not routinely available. LKE– blood is not available for transfusion; thus, "serologically least incompatible" blood is given.

Unnamed Blood Group Collection: Anti-Le^c and Anti-Le^d

Despite their name, the determinants recognized by these antibodies are not the product of transferases encoded by Lewis (*FUT3*) genes. However, like Lewis antigens, Le^c and Le^d antigens are adsorbed onto RBCs. Anti-Le^c reacts with an antigen expressed on Le(a–b–) RBCs from adult non-secretors, whereas anti-Le^d reacts with an antigen expressed on Le(a–b–) RBCs from adult secretors. These antibodies have no clinical significance with regard to transfusion.

MN CHO Collection

The antigens of the MN CHO collection (Table 1) are expressed on glycophorin A (GPA) molecules that have altered levels of glycosylation in that the amount of *N*-acetyl neuraminic acid or *N*-acetyl-D-glucosamine is different from that present on "conventional" GPA. Some antigens are associated with altered GPA^N, whereas others are associated with altered GPA^M. The antibodies that detect these determinants are considered to be non–red cell immune and of no clinical significance, but can complicate antibody identification. The optimal reactivity of these antibodies is at room temperature, and most are likely to be IgM, although available information is limited. For readers wishing to learn more about these antigens, please refer to Daniels,[3] Issitt and Anstee,[7] or Dahr et al.[8]

Low-Incidence Antigens (ISBT 700 Series)

Antigens in the 700 series occur with an incidence of less than 1 percent in most populations studied and do not have the criteria to be included in an established blood group system or collection. Inheritance of the antigen through at least two generations must be demonstrated for the antigen to join the 700 series.[2] In the clinical setting, antibodies to antigens in the 700 series are usually found because the antibody has caused HDFN or a transfusion reaction if an electronic crossmatch was used. These antibodies may also be found when a patient's serum or plasma reacts with a single RBC sample during compatibility testing. Before the introduction of monoclonal blood typing reagents, many antibodies to low-incidence antigens were found as contaminants in blood typing reagents. Currently, there are 17 antigens in the 700 series. Some of the antibodies to these antigens are red cell immune, whereas others appear to be "naturally occurring" (or non–red cell immune). Antibodies to low-incidence antigens rarely cause a problem for transfusion as compatible blood is readily available. Several antibodies have caused HDFN, but this is a rare occurrence because of the low prevalence of these antigens that may have been identified in only one family; they are summarized in Table 2.

High-Incidence Antigens (ISBT 901 Series)

Antigens in the 901 series occur with an incidence of greater than 90 percent in most populations studied and cannot be included in an established blood group system or collection. This series was originally numbered as the 900 series but was renumbered as the 901 series in 1988 after many of the antigens were relocated to systems or collections.[9] To be included in this series, it must be demonstrated that the antigen is lacking from the RBCs of at least two siblings (i.e., that the antigen-negative phenotype is genetically determined).

Table 2. Antigens and antibodies of the ISBT 700 series of low-incidence antigens

			Antibody characteristics*			
ISBT number	Name	Symbol	Immune	Stimulus unknown	Caused HDFN†	Number of antibodies found‡
700002	Batty	By	Yes	Yes	DAT+	Many
700003	Christiansen	Chr^a	…	Yes	No	Few
700005	Biles	Bi	Yes	…	Probably	Few
700006	Box	Bx^a	…	Yes	…	Few
700017	Torkildsen	To^a	…	Yes	…	Many
700018	Peters	Pt^a	…	Yes	…	Many
700019	Reid	Re^a	Yes	…	Mild	Few
700021	Jensen	Je^a	…	Yes	No	Few
700028	Livesay	Li^a	Yes	Yes	Mild	Few
700039	Milne	…	…	Yes	…	Many
700040	Rasmussen	RASM	Yes	…	DAT+	1
700044	…	JFV	Yes	…	DAT+ to moderate	Few
700045	Katagiri	Kg	Yes	…	Severe†	1
700047	Jones	JONES	Yes	…	Moderate	Few
700049	…	HJK	Yes	…	Severe†	1
700050	…	HOFM	Yes	…	Mild	1
700054	…	REIT	Yes	…	Severe†	1

*The antibodies in this table may occur as IgM, IgG, or a mix of both.

†Severe HDFN requiring intrauterine or exchange transfusion.

‡Few = 1–5 examples; many = >13 examples.

ISBT = International Society of Blood Transfusion; HDFN = hemolytic disease of the fetus and newborn; DAT = direct antiglobulin test.

Currently, there are six antigens in the 901 series, and they are shown in Table 3.

Anti-Sd^a

Anti-Sd^a detects an antigen with a prevalence of about 91 percent. The strength of Sd^a antigen expression is variable, from barely detectable, such that some RBCs may erroneously be considered as Sd(a–), to high levels of Sd^a expression [termed Sd(a++)] that results in non-group A_1 RBCs being agglutinated by Dolichos lectin. Anti-Sd^a is easily recognized because it causes a characteristic mixed-field agglutination of orange refractile agglutinates that may look like bunches of grapes when viewed microscopically. Urine (human or guinea pig) that contains Tamm-Horsfall glycoprotein can be used to inhibit anti-Sd^a. The antibodies are mostly IgM, but some are IgG; they often react at lower temperatures and by antiglobulin methods. Although anti-Sd^a is not generally considered a transfusion hazard, and Sd(a–) RBCs are not required for transfusion, serologically least incompatible RBCs should be selected to avoid Sd(a++) RBCs.

Table 3. Antigens and antibodies of the ISBT 901 series of high-incidence antigens

ISBT number	Name	Symbol	Clinical significance of antibody
901008	…	Emm	See text
901009	Anton	AnWj	See text
901012	Sid	Sd^a	Not considered to be (may increase titer) but two suspected HTRs associated with RBCs with unusually strong expression of Sda [Sd(a++)]
901014	…	PEL	The four makers of anti-PEL (or the related specificity anti-MTP) were transfused; three were pregnant; no indication of HDFN; normal RBC survival with one anti-PEL but potential reduced survival with anti-MTP
901015	…	ABTI	No evidence of HDFN; no data for transfusion reactions; first three probands were Israeli
901016	…	MAM	See text

ISBT = International Society of Blood Transfusion; HTR = hemolytic transfusion reaction; RBCs = red blood cells; HDFN = hemolytic disease of the fetus and newborn.

Anti-Emm

The Emm antigen is carried on a glycosylphosphatidylinositol-linked protein in the RBC membrane. Because the Emm– phenotype is rare, it follows that few examples of the antibody have been reported. IgG anti-Emm are more common than IgM (four out of five) and, although the original anti-Emm reacted in direct testing at 4°C, most of these antibodies react optimally by antiglobulin methods. Some anti-Emm have bound complement. There is no information regarding HDFN as six of the reported anti-Emm were found in untransfused males. Until 2013, anti-Emm had not been implicated in a transfusion reaction, possibly because the patients were not transfused.

In 2013, Takahashi et al.[10] reported the case of a 58-year-old man who had never been transfused but who was in urgent need of transfusion because of massive bleeding from an abdominal stab wound. Pretransfusion testing demonstrated an antibody that was reactive with all panel cells but nonreactive with the autologous RBCs. Thirty minutes after transfusion of a crossmatch-incompatible unit of blood, the patient experienced a drop in blood pressure and hematuria. Because his hemoglobin was 5.5 g/dL, another 2 units of blood were transfused, and his vital signs became stable. A transfusion on the third day was uneventful. On day 6, after receiving 30 mL blood, the patient vomited, had cola-colored urine (total bilirubin 6.1 mg/dL, lactate dehydrogenase LDH 912 U/L), and the transfusion was stopped. No further transfusions were administered, and the patient recovered with iron therapy. After transfusion, his RBCs reacted in the DAT: 1+ on day 1, 2+ on day 3, and negative on day 7 (the day after the acute HTR occurred), suggesting that no transfused RBCs remained in circulation. The antibody in the patient's plasma reacted in saline at 4°C (2+), by albumin–indirect antiglobulin test (IAT) (2+), polyethylene glycol-IAT (2+), and papain-IAT (3+), and was identified as anti-Emm. The antibody had IgG1 and IgG3 components and was shown to fix complement. Before transfusion, the antibody titer was 16 by saline-IAT and rose to 128 by day 10.

Anti-AnWj

Anti-AnWj has been an intriguing specificity from the beginning and can at times be tricky to identify. An apparent alloantibody to an antigen called Anton, which was thought to be an antigen in the Lutheran system, was reported in 1982. A year later, an autoantibody called anti-Wj was reported, and in 1985, it was shown that both antibodies detected the same antigen (now named AnWj).[4] The AnWj antigen is absent or only weakly expressed on RBCs of the dominant Lu(a–b–) phenotype encoded by *In(Lu)*. Lu(a–b–) dominant-type RBCs not only have greatly reduced expression of Lutheran antigens, but also of AnWj, Indian, Knops, Cs^a, MER2, and P1 blood group antigens.[3,4] This antigen suppression is the result of heterozygosity for any one of several inactivating changes in *KLF1,* the gene that encodes erythroid Krüppel-like factor (EKLF). These changes cause a general reduction of transcription of several erythroid genes and reduced levels of gene products such as the Lu glycoprotein. Therefore, if a patient's plasma does not react with RBCs of the dominant Lu(a–b–) phenotype, it cannot be assumed that the reactivity is directed at a Lutheran antigen. Furthermore, antibody identification may also be complicated by the fact that the level of suppression on dominant-type Lu(a–b–) RBCs varies so that some RBC samples may react weakly with some examples of anti-AnWj.

Antibodies with AnWj specificity are predominantly autoantibodies but may appear as an alloantibody because of transient (often long-term) suppression of AnWj on the RBCs of the antibody maker. The antibody is frequently found in association with lymphoma (Hodgkin's and non-Hodgkin's) and other lymphoid malignancies, immunologic disorders, and autoimmune hemolytic anemia. The only two reported examples of alloanti-AnWj were found in two Arab Israeli sisters with a history of multiple pregnancies but no transfusions.[11] Anti-AnWj has not been associated with HDFN, however. The fact that pregnancy appeared to be the immunizing event for the sisters is somewhat puzzling, since fetal cells express almost no AnWj antigen and RBCs from cord samples type AnWj–.

Some patients with anti-AnWj tolerate "random" units,[12] whereas others do not and require blood with the dominant Lu(a–b–) phenotype. The antibody has been implicated in severe HTRs, and its hemolytic potential was substantiated by monocyte monolayer assay (MMA) or in vivo RBC survival studies.[3,13] Xu et al.,[14] in an informative review article, described the case of a 56-year-old woman with aplastic anemia with anti-AnWj identified in her plasma. Transfusion of "random" units resulted in an acute HTR. The anti-AnWj activated complement, and only C3 was detected on the patient's RBCs when tested in the DAT. Blood from donors with the dominant Lu(a–b–) phenotype was well tolerated. There is a limited supply of blood with this rare phenotype, however, and long-term provision can be difficult, if not impossible. Similar to the case described by Xu et al.,[14] a patient with anti-AnWj and a diagnosis of acute myeloid leukemia was transfused with

a "random" unit, matched for common antigens. About 100 mL was transfused before the procedure was stopped. The patient suffered from an HTR, and a follow-up MMA at a later date showed that the anti-AnWj was expected to be clinically significant. The patient's RBCs, when tested in the DAT, were coated with C3 only, and an eluate prepared from the RBCs was nonreactive. Attempts to reduce the anti-AnWj production by giving rituximab were not successful in this case.

Because dominant type Lu(a–b–) donors are rare, it is important to determine if a patient with anti-AnWj can or cannot tolerate blood that is not AnWj–. The tiny amount of AnWj on dominant type Lu(a–b–) RBCs does not appear to affect RBC survival. RBCs with the very rare recessive or X-linked Lu(a–b–) phenotypes fully express AnWj and should not be used for patients with anti-AnWj (in the unlikely event that these rare types are available).[4]

Anti-MAM

Anti-MAM is an extremely rare antibody, and the MAM antigen has a high incidence (greater than 99%) in all populations. The antibody was first reported in 1993[15] and assigned to the 901 series of high-incidence antigens in 1999. MAM antigen is expressed on cord RBCs, lymphocytes, granulocytes, monocytes, and probably on platelets.

Four of the five reported examples of anti-MAM were identified during pregnancy in mothers who had never been transfused. One of the mothers was of Irish and Cherokee descent, and the four other antibody makers were all of Arab descent. The anti-MAM were IgG antibodies; antibodies from two patients were subtyped as IgG1 and IgG3, and one also had an IgG2 component. Anti-MAM has been associated with clinical HDFN and/or thrombocytopenia. Of the four babies born to mothers with anti-MAM, the course of each pregnancy was slightly different, although the RBCs of the newborns all were reactive in the DAT. One baby had no clinical HDFN but experienced fetal and neonatal thrombocytopenia and required platelet transfusion. Another had severe HDFN and required intrauterine RBC transfusion. In addition, this baby presented with fetal and neonatal thrombocytopenia, but platelet transfusion was not required. A third baby had mild HDFN requiring ultraviolet phototherapy because of elevated bilirubin levels but no fetal or neonatal thrombocytopenia. A fourth baby required no RBC transfusion, platelets, or phototherapy. The platelet count of the fourth baby was slightly reduced (132,000/μL [normal range 150,000–450,000/μL]), but by 8 weeks of age, the count had increased to 305,000/μL.[16]

With regard to transfusion, the results of the MMA suggest that anti-MAM has the potential to substantially reduce the survival of transfused MAM+ RBCs.[17,18] Ideally, MAM– RBCs should be transfused, but, because of their extreme rarity, cautious transfusion of serologically "least incompatible" RBCs may be the only option.

Conclusions

The antibodies made to some antigens in the ISBT blood group collections, 700 series, and 901 series have established and documented clinical significance, either for transfusion or for HDFN, but for others the information is limited. When antigen-negative blood is not available and information regarding the clinical significance of an antibody is lacking, it is best to proceed with caution, since any antibody reactive by IAT may have clinical potential. At the same time, the risk of not transfusing versus a potential transfusion reaction must be considered, since a patient is more likely to die from lack of blood than from a transfusion reaction. Clinical significance is mostly based on previous experience with a particular specificity. Thus, to predict clinical significance, the antibody specificity must be correctly identified. At times, this can be a challenge. In vitro tests such as the MMA may also be informative.

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